Listing of Claims

1. (Currently Amended) A method for the treatment of ~~a *Flaviviridae* virus infection or~~ a human immunodeficiency virus (HIV) infection, comprising administration, to a subject in need thereof, of a therapeutically effective amount of an inhibitor of a *src* family kinase, whereby the ~~*Flavivirida* virus infection or~~ human immunodeficiency virus (HIV) infection is diminished relative to a non-treated subject.

2-4 (Canceled)

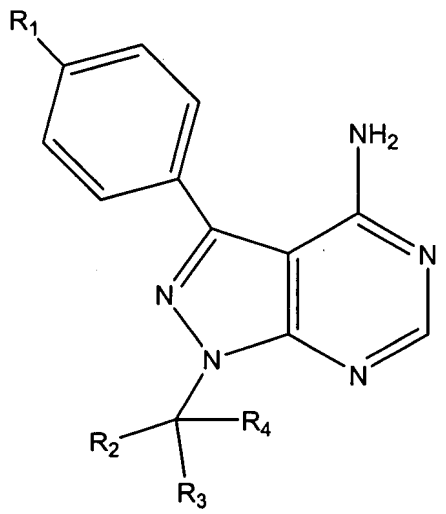
5. (Original) The method of claim 1, wherein the *src* family kinase is c-yes kinase.

6. (Original) The method of claim 1, wherein the inhibitor comprises a *src* family kinase-specific antisense oligonucleotide.

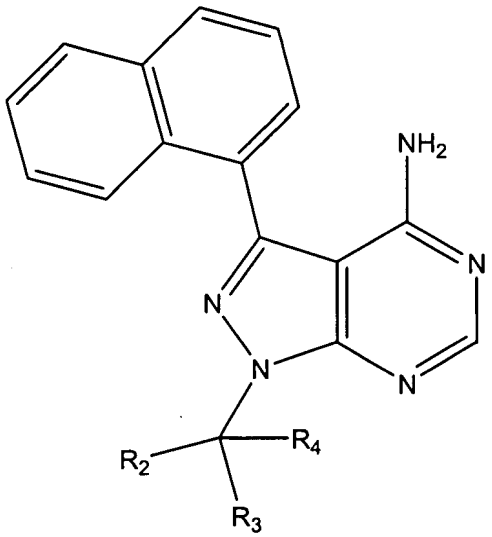
7. (Original) The method of claim 6, wherein the antisense oligonucleotide is a phosphorodiamidate morpholino oligomer (PMO).

8. (Original) The method of claim 1, wherein the inhibitor comprises *src* family kinase-specific siRNA.

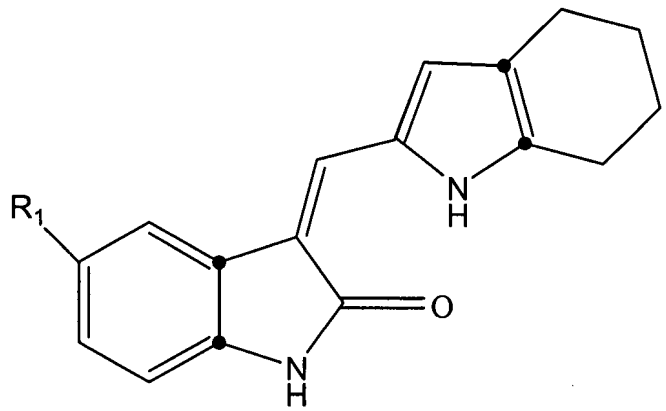
9. (Original) The method of claim 1, wherein the inhibitor comprises a small molecule inhibitor of a *src* family kinase, wherein the small molecule inhibitor is a molecule, or a pharmaceutically acceptable salt thereof, selected from the Formula group consisting of Formula I, Formula I(b), Formula II, Formula III, Formula IV and Formula V:



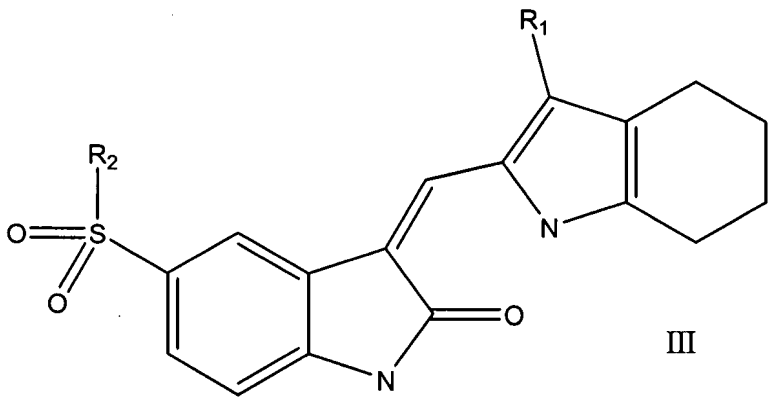
Formula I



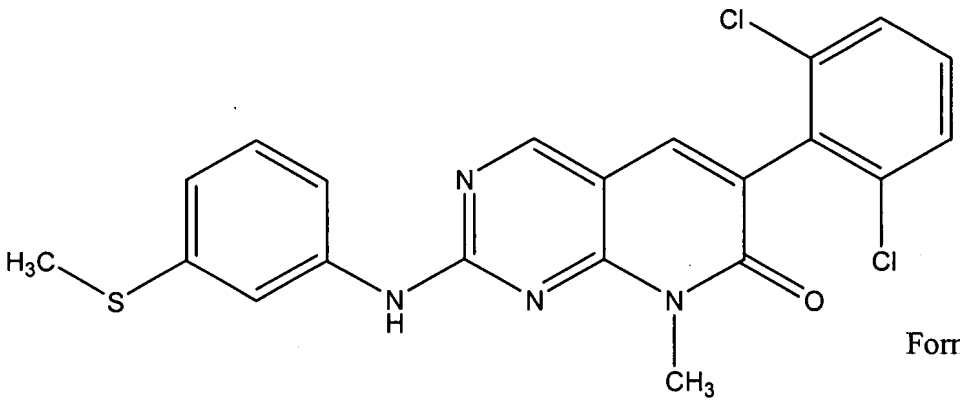
I(b)



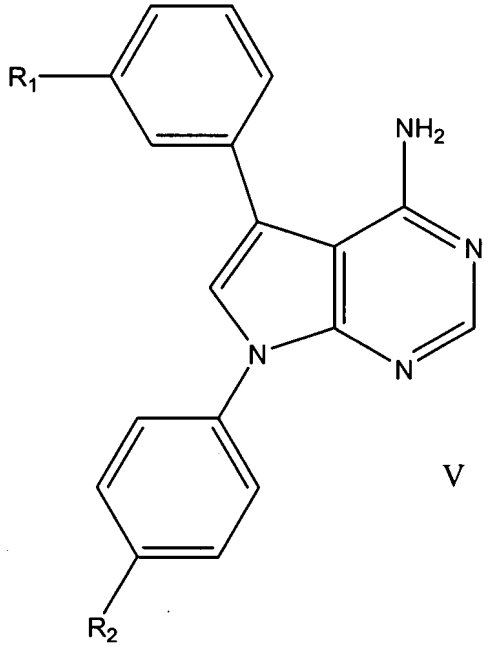
Formula II



III



Formula IV



V

wherein for Formula I or I(b), R_1 is halogen or methyl, and R_2, R_3 and R_4 are independently a C1-C3 straight or branched alkyl; wherein for Formula II, R_1 is $-SO_2N(CH_3)_2$, or $-SO_2NH_2$; wherein for Formula III, R_2 is C_2H_5 or NHR_3, wherein R_3 is a C1 to C3 linear or branched alkyl

moiety, and wherein R_1 is independently $-(CH_2)_3N(CH_3)_2$, $-CH_2N(CH_2CH_2)_2O$, $-(CH_2)_2N(CH_2CH_2)_2O$, $-(CH_2)_3N(CH_2CH_2)_2O$, or $-(CH_2)_3N(CH_2CH_2)_2N\ CH_3$; and wherein for Formula V, R_1 is either H or $-OCH_3$, wherein R_2 is independently $-(CH_2)_2OH$, $-CH_2COOH$, $-(CH_2)_2N(CH_3)_2$, $-(CH_2)_2NH(CH_2)_2OH$, $-(CH_2)_2NCH_3(CH_2)_2OCH_3$, $-(CH_2)_2N(CH_2CH_2)_2NCH_3$, or $-(CH_2)_2N(CH_2CH_2)_2CHOH$.

10. (Original) The method of claim 9, wherein according to Formula I, the small molecule inhibitor is 4-Amino-5-(4-chlorophenyl)-7-(t-butyl)pyrazolo[3,4-d]pyrimidine ("PP2").

11. (Original) The method of claim 9, wherein according to Formula I(b), the small molecule inhibitor is 4-Amino-1-*tert*-butyl-3-(1'-naphthyl)pyrazolo[3,4-d]pyrimidine.

12. (Original) The method of claim 9, wherein according to Formula II, the small molecule inhibitor is 2-oxo-3-(4,5,6,7-tetrahydro-1*H*-indol-2-ylmethylene)-2,3-dihydro-1*H*-indole-5-sulfonic acid dimethylamide.

13. (Original) The method of claim 9, wherein according to Formula III, R_1 is: $-(CH_2)_3N(CH_3)_2$; $-(CH_2)_3N(CH_2CH_2)_2O$; or $-(CH_2)_3N(CH_2CH_2)_2NCH_3$.

14. (Original) The method of claim 9, wherein according to Formula III, R_2 is NH CH_3 and R_1 is $-(CH_2)_3N(CH_3)_2$, wherein the small molecule inhibitor is 3-[3-(3-dimethylamino-propyl)-4,5,6,7-tetrahydro-1*H*-indol-2-ylmethylene]-2-oxo-2,3-dyhydro-1*H*-indole-5-sulphonic acid methylamide.

15. (Original) The method of claim 9, wherein according to Formula III, R_2 is C_2H_5, and R_1 is $-(CH_2)_3N(CH_3)_2$.

16. (Original) The method of claim 9, wherein according to Formula III, R_2 is NH CH_3 and R_1 is $-(CH_2)_3N(CH_2CH_2)_2O$.

17. (Original) The method of claim 9, wherein according to Formula III, R_2 is NH CH_3 and R_1 is $-(CH_2)_3N(CH_2CH_2)_2N\ CH_3$.

18. (Original) The method of claim 9, wherein according to Formula III, R_2 is C_2H_5, and R_1 is $-(CH_2)_3N(CH_2CH_2)_2N\ CH_3$.

19. (Original) The method of claim 9, wherein according to Formula V, R_1 is $-OCH_3R_2$, and R_2 is $-(CH_2)_2N(CH_2CH_2)_2CHOH$.

20- 34. (Canceled).

35. (Currently Amended) A method for identification of an agent having therapeutic utility for the treatment of a *Flaviviridae* virus infection or human immunodeficiency virus infection, comprising:

-obtaining cells suitable to support a *Flaviviridae* virus or a human immunodeficiency virus (HIV) infection;

-infecting the cells with the *Flaviviridae* virus or human immunodeficiency virus;

-contacting the infected cells with an agent that inhibits a src family kinase; and

-determining whether the *Flaviviridae* virus infection or human immunodeficiency virus infection is diminished relative to control infected cells not contacted by the agent, thereby ~~idenfying~~identifying the agent as having therapeutic utility for the treatment of the Flaviviridae virus infection or human immunodeficiency virus (HIV) infection.

36. (Original) The method of claim 35, wherein the *src* family kinase is c-yes kinase.

37. (Original) The method of claim 35, wherein the *Flaviviridae* virus is selected from the group consisting of a flavivirus and hepatitis C virus (HCV).

38. (Original) The method of claim 35, wherein the flavivirus is selected from the group consisting of West Nile virus (WNV), Japanese encephalitis virus (JEV), yellow fever virus (YFV), and Dengue fever virus (DEN).

39. (Original) The method of claim 35, wherein the *Flaviviridae* virus is hepatitis C virus (HCV).

40. (Original) The method of claim 35, wherein the inhibitor comprises a *src* family kinase-specific antisense oligonucleotide.

41. (Original) The method of claim 40, wherein the antisense oligonucleotide is a phosphorodiamidate morpholino oligomer (PMO).

42. (Original) The method of claim 35, wherein the inhibitor comprises *src* family kinase-specific siRNA.

43. (Original) The method of claim 35, wherein the inhibitor comprises a small molecule inhibitor of a *src* family kinase, wherein the small molecule inhibitor is a molecule, or a pharmaceutically acceptable salt thereof, selected from the Formula group consisting of Formula I, Formula I(b), Formula II, Formula III, Formula IV and Formula V, all according to claim 8.

44. (Original) The method of claim 35, wherein the cells suitable to support flavivirus infection are selected from the group consisting of primary human hepatocellular carcinoma derived cells or cell-lines derived therefrom, Huh 7 cells, neuroblastoma cells or cell-lines derived therefrom, SKN-MC cells, and combinations thereof.

45. (Original) The method of claim 35, wherein infection precedes contacting of the cells with the agent.

46. (Original) The method of claim 35, wherein infection is subsequent to contacting of the cells with the agent.

Claims 47-79 (Canceled) .

80. (Previously Presented) The method of claim 35, wherein the method identifies an agent having therapeutic utility for the treatment of an human immunodeficiency virus infection, and wherein the cells suitable to support human immunodeficiency virus (HIV) infection are selected from the group consisting of myeloid cells, or T-cells, and combinations thereof.

81. (Original) The method of claim 80, wherein the cells are of the myeloid cell line THP-1.

82. (Original) The method of claim 80, wherein the cells are of the T-cell leukemia cell line MT-2.

83. (Previously Presented) The method of claim 80, wherein infection precedes contacting of the cells with the agent.

84. (Previously Presented) The method of claim 80, wherein infection is subsequent to contacting of the cells with the agent.

85. (Previously Presented) A method for the treatment of a human immunodeficiency virus (HIV) infection or related condition, comprising administration, to a subject in need thereof, of a therapeutically effective amount of an inhibitor of a human immunodeficiency virus-induced cellular gene sequence selected from the group consisting of HMG20B, HRH1, NP, c-YES, corresponding to SEQ ID NOS:1-9, and combinations thereof, whereby the human immunodeficiency virus (HIV) infection or related condition is diminished, at least to some extent, relative to a non-treated subject.

86. (Previously Presented) The method of claim 85, wherein the human immunodeficiency virus-induced cellular gene sequence is that of HMG20, corresponding to SEQ ID NOS:1-2.

87. (Previously Presented) The method of claim 85, wherein the human immunodeficiency virus-induced cellular gene sequence is that of HRH1, corresponding to SEQ ID NOS:3-5.

88. (Previously Presented) The method of claim 85, wherein the human immunodeficiency virus-induced cellular gene sequence is that of NP, corresponding to SEQ ID NOS:6-7.

89. (Previously Presented) The method of claim 85, wherein the human immunodeficiency virus-induced cellular gene sequence is that of c-YES, corresponding to SEQ ID NOS:8-9.

90. (Previously Presented) The method of claim 85, wherein the inhibitor comprises

a antisense oligonucleotide specific for the respective human immunodeficiency virus-induced cellular gene sequence.

91. (Original) The method of claim 90, wherein the antisense oligonucleotide is a phosphorodiamidate morpholino oligomer (PMO).

92. (Previously Presented) The method of claim 85, wherein the inhibitor comprises siRNA specific for the respective human immunodeficiency virus-induced cellular gene sequence.

93. (Previously Presented) The method of claim 85, wherein the inhibitor comprises a small molecule inhibitor specific for the respective human immunodeficiency virus-induced cellular gene sequence.

94-102 (Canceled).

103. (Previously Presented) A method for identification of an agent having therapeutic utility for the treatment of a human immunodeficiency virus (HIV) infection or related condition, comprising:

-obtaining cells suitable to support a human immunodeficiency virus (HIV) infection;

-infecting the cells with the human immunodeficiency virus (HIV);

-contacting the infected cells with an agent that inhibits a human immunodeficiency virus-induced cellular gene sequence selected from the group consisting of HMG20B, HRH1, NP, c-YES, corresponding to SEQ ID NOS:1-9, and combinations thereof; and

-determining whether the human immunodeficiency virus (HIV) infection is diminished relative to control infected cells not contacted by the agent, whereby the therapeutic agent is identified.

104. (Previously Presented) The method of claim 103, wherein the human immunodeficiency virus-induced cellular gene sequence is that of HMG20, corresponding to

SEQ ID NOS:1-2.

105. (Previously Presented) The method of claim 103, wherein the human immunodeficiency virus-induced cellular gene sequence is that of HRH1, corresponding to SEQ ID NOS:3-5.

106. (Previously Presented) The method of claim 103, wherein the human immunodeficiency virus-induced cellular gene sequence is that of NP, corresponding to SEQ ID NOS:6-7.

107. (Previously Presented) The method of claim 103, wherein the human immunodeficiency virus-induced cellular gene sequence is that of c-YES, corresponding to SEQ ID NOS:8-9.

108. (Previously Presented) The method of claim 103, wherein the inhibitor comprises a antisense oligonucleotide specific for the respective human immunodeficiency virus-induced cellular gene sequence.

109. (Original) The method of claim 108, wherein the antisense oligonucleotide is a phosphorodiamidate morpholino oligomer (PMO).

110. (Previously Presented) The method of claim 103, wherein the inhibitor comprises siRNA specific for the respective human immunodeficiency virus-induced cellular gene sequence.

111. (Previously Presented) The method of claim 103, wherein the inhibitor comprises a small molecule inhibitor specific for the respective human immunodeficiency virus-induced cellular gene sequence.

112. (Original) The method of claim 103, wherein the cells suitable to support human immunodeficiency virus (HIV) infection are selected from the group consisting of myeloid cells, or T-cells, and combinations thereof.

113. (Original) The method of claim 103, wherein the cells are of the myeloid cell line THP-1.

114. (Original) The method of claim 103, wherein the cells are of the T-cell leukemia cell line MT-2.

115. (Original) The method of claim 103, wherein infection precedes contacting of the cells with the agent.

116. (Original) The method of claim 103, wherein infection is subsequent to contacting of the cells with the agent.

117. (New) The method of claim 9, wherein the small molecule inhibitor inhibits replication of the human immunodeficiency virus.

118. (New) A method for inhibiting replication of human immunodeficiency virus, comprising

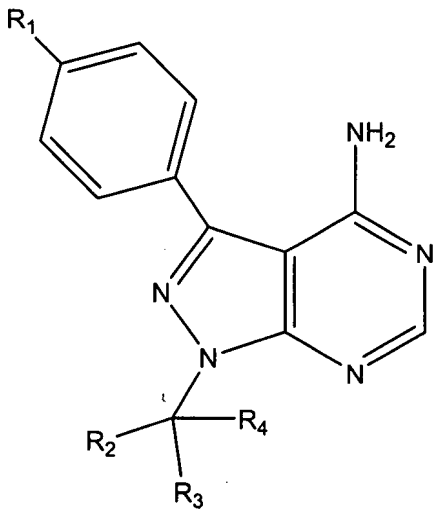
contacting a cell infected with the human immunodeficiency virus with a therapeutically effective amount of a c-yes inhibitor,

thereby decreasing the replication of the human immunodeficiency virus.

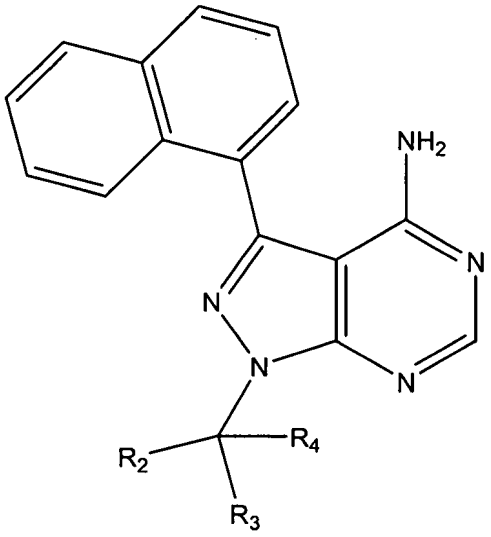
119. (New) The method of claim 118, wherein the c-yes inhibitor is a small molecule inhibitor of c-yes.

120. (New) The method of claim 119, wherein the cell is *in vivo*.

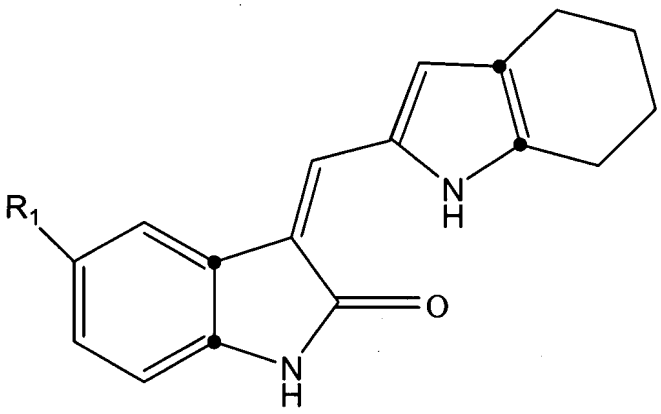
121. (New) The method of claim 119, wherein the inhibitor comprises a small molecule or a pharmaceutically acceptable salt thereof, selected from the Formula group consisting of Formula I, Formula I(b), Formula II, Formula III, Formula IV and Formula V:



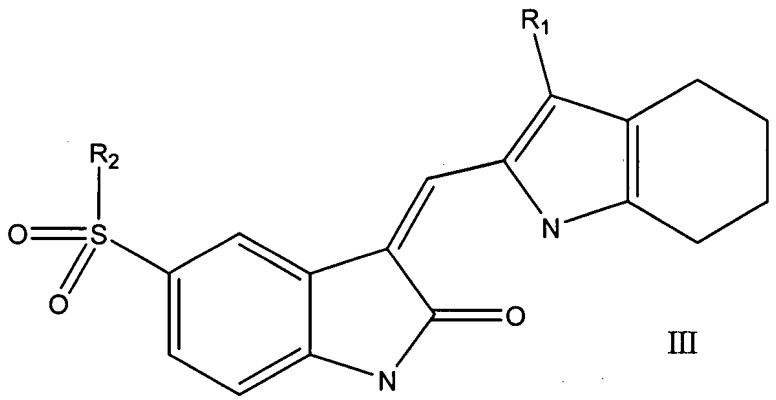
Formula I



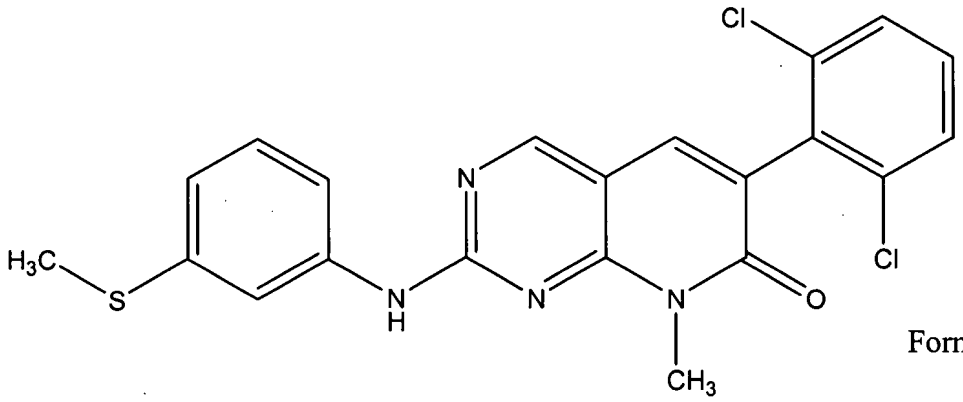
I(b)



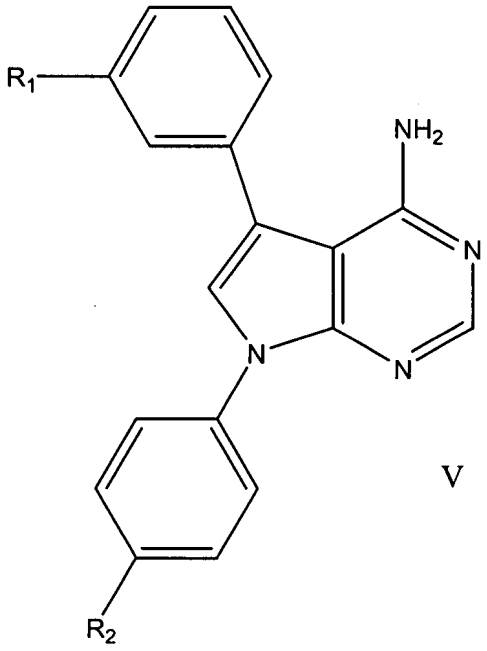
Formula II



III



Formula IV



V

wherein for Formula I or I(b), R_1 is halogen or methyl, and R_2, R_3 and R_4 are independently a C1-C3 straight or branched alkyl; wherein for Formula II, R_1 is $-SO_2N(CH_3)_2$, or $-SO_2NH_2$; wherein for Formula III, R_2 is C_2H_5 or NHR_3, wherein R_3 is a C1 to C3 linear or branched alkyl

moiety, and wherein R_1 is independently $-(CH_2)_3N(CH_3)_2$, $-CH_2N(CH_2CH_2)_2O$, $-(CH_2)_2N(CH_2CH_2)_2O$, $-(CH_2)_3N(CH_2CH_2)_2O$, or $-(CH_2)_3N(CH_2CH_2)_2N\ CH_3$; and wherein for Formula V, R_1 is either H or $-OCH_3$, wherein R_2 is independently $-(CH_2)_2OH$, $-CH_2COOH$, $-(CH_2)_2N(CH_3)_2$, $-(CH_2)_2NH(CH_2)_2OH$, $-(CH_2)_2NCH_3(CH_2)_2OCH_3$, $-(CH_2)_2N(CH_2CH_2)_2NCH_3$, or $-(CH_2)_2N(CH_2CH_2)_2CHOH$.

122. (New) The method of claim 121, wherein according to Formula I, the small molecule inhibitor is 4-Amino-5-(4-chlorophenyl)-7-(t-butyl)pyrazolo[3,4-d]pyrimidine ("PP2").

123. (New) The method of claim 121, wherein according to Formula I(b), the small molecule inhibitor is 4-Amino-1-*tert*-butyl-3-(1'-naphthyl)pyrazolo[3,4-d]pyrimidine.

124. (New) The method of claim 121, wherein according to Formula II, the small molecule inhibitor is 2-oxo-3-(4,5,6,7-tetrahydro-1*H*-indol-2-ylmethylene)-2,3-dihydro-1*H*-indole-5-sulfonic acid dimethylamide.

125. (New) The method of claim 121, wherein according to Formula III, R_1 is: $-(CH_2)_3N(CH_3)_2$; $-(CH_2)_3N(CH_2CH_2)_2O$; or $-(CH_2)_3N(CH_2CH_2)_2NCH_3$.

126. (New) The method of claim 121, wherein according to Formula III, R_2 is NH CH_3 and R_1 is $-(CH_2)_3N(CH_3)_2$, wherein the small molecule inhibitor is 3-[3-(3-dimethylamino-propyl)-4,5,6,7-tetrahydro-1*H*-indol-2-ylmethylene]-2-oxo-2,3-dyhydro-1*H*-indole-5-sulphonic acid methylamide.

127. (New) The method of claim 121, wherein according to Formula III, R_2 is C_2H_5, and R_1 is $-(CH_2)_3N(CH_3)_2$.

128. (New) The method of claim 121, wherein according to Formula III, R_2 is NH CH_3 and R_1 is $-(CH_2)_3N(CH_2CH_2)_2O$.

129. (New) The method of claim 121, wherein according to Formula III, R_2 is NH CH_3 and R_1 is $-(CH_2)_3N(CH_2CH_2)_2N\ CH_3$.

130. (New) The method of claim 121, wherein according to Formula III, R_2 is C_2H_5, and R_1 is $-(CH_2)_3N(CH_2CH_2)_2N\ CH_3$.

131. (New) The method of claim 121, wherein according to Formula V, R_1 is $-OCH_3R_2$, and R_2 is $-(CH_2)_2N(CH_2CH_2)_2CHOH$.